Carotid endarterectomy in patients less than 50 years old

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Purpose: **The purpose of this study was to compare the results of carotid endarterectomy (CEA) in a young population with premature atherosclerosis with the results of an older control group, examining perioperative morbidity and mortality data, recurrent stenosis and symptoms, late stroke, and survival data.**

Methods: **We retrospectively studied 26 patients less than 50 years old (mean, 43.2 ± 3.8 years) and 30 patients greater than 55 years old (mean, 69.1 ± 7.4 years) who underwent CEA during the same time period. Data were obtained regarding demographics, atherosclerotic risk factors, indication for CEA, perioperative complications, recurrent stenosis and symptoms, late stroke, and survival.**

Results: **Smoking was more prevalent among young patients who underwent CEA (92% vs 70%; $p = 0.036$). Young patients were also more likely to be symptomatic at presentation (92% vs 57%; $p = 0.003$). The perioperative mortality rate (0% vs 0%) and neurologic morbidity rate (0% vs 3%; $p = 1.000$) were low for the study patients. During a mean follow-up of 67 ± 42.7 months, there was no significant difference in survival rate (5-year survival rate, 93% vs 81%; $p = 0.373$), rate of late ipsilateral (4% vs 3%) and contralateral (4% vs 3%) stroke, restenosis and occlusion (26.9% vs 14.3%), recurrent symptoms (22% vs 17%), reoperation (11.5% vs 5.7%), or contralateral disease (17% vs 23%) development that required surgery for the study or the control cohorts.**

Conclusions: **Our data show that there is a high incidence of smoking and symptomatic presentation among young patients in whom carotid occlusive disease develops. CEA may be performed in young patients with low perioperative morbidity and mortality rates. Recurrent disease, late stroke, and survival rates are not significantly different than for older patients. Follow-up with serial duplex ultrasound and reoperation for symptomatic and high-grade asymptomatic restenosis may decrease the risk of late stroke. (J Vasc Surg 1997;26:447-55.)**

Recent multicenter trials (NASCET and ACAS)[1,2] have established that carotid endarterectomy (CEA) is superior to medical treatment in the prevention of strokes. Although cerebrovascular disease generally affects patients in their seventh and eighth decades of life, a population has been identified that is affected by the premature development of atherosclerosis and carotid occlusive disease. This premature form of atherosclerosis is uncommon and may be more virulent and diffuse than that seen in older populations. Series report decreased longevity[3,4] and increased risk for recurrent disease[5,6] after surgery for this younger population. In this study, we reviewed our 15-year experience with CEA in patients less than 50 years old and compared results for recurrent disease, late stroke, and survival with results for older patients who underwent CEA.

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PATIENTS AND METHODS

We retrospectively reviewed the records of patients less than 50 years old with carotid occlusive disease who underwent CEA by our practice from 1981 to 1996. CEA was performed in 4091 patients of all ages during this period. Twenty-six patients (0.6%) less than 50 years old underwent 33 primary CEAs, including staged bilateral CEAs in three patients and eventual contralateral CEA in four patients. In addition, three patients underwent second-

Table I. Demographics, risk factors, and neurologic presentation

	Patients (n = 26)	*Control (n = 30)*	*p*
Mean age (yr)	43.2 ± 3.8	69.1 ± 7.4	
Female	11 (42%)	17 (57%)	0.284
Neurologic presentation			
Symptomatic	24 (92%)	17 (57%)	0.003
TIA	18 (69%)	15 (50%)	0.145
CVA	6 (23%)	2 (7%)	0.080
Risk factors			
Smoking	24 (92%)	21 (70%)	0.036
Hypertension	13 (50%)	21 (70%)	0.126
Hyperlipidemia	8 (31%)	6 (20%)	0.353
Diabetes	5 (19%)	6 (20%)	0.942
CAD	8 (31%)	15 (50%)	0.246

TIA, Transient ischemic attack; *CVA,* cerebrovascular accident; *CAD,* coronary artery disease.

ary procedures for recurrent carotid stenosis during the study period. Hospital and outpatient records were reviewed to obtain demographics and information concerning risk factors for atherosclerosis, neurologic presentation, preoperative radiologic evaluation, operative technique, and postoperative course, including additional operations.

Data concerning long-term outcome after CEA were obtained from hospital and outpatient records and by phone survey. After the operation, all patients were followed-up with routine outpatient examinations and duplex ultrasound (DUS) surveillance. In general, clinical follow-up was performed at 6-month intervals, with DUS obtained at 6 and 12 months after the operation and then yearly if patients were asymptomatic and there was no evidence of significant restenosis. Carotid stenosis was determined using standardized velocity criteria. Restenosis (luminal diameter reduction) <40% was considered mild, restenosis of 40% to 69% was considered moderate (significant), and restenosis ≥70% was considered high-grade. Patients who had recurrent neurologic symptoms or evidence of high-grade restenosis by DUS were studied with cerebral arteriography.

For comparison, a control group of 30 patients aged 55 and older (age 55 to 78 years; mean, 69.1 ± 7.4 years) were randomly selected from the group of 4091 patients who underwent CEA during the study period. Patients aged 50 to 54 years were excluded to avoid age overlap. This control group underwent 39 CEAs, performed by the same surgeons, during the same 15-year study interval. In this cohort, two patients underwent staged bilateral CEAs and seven patients underwent eventual contralateral CEAs. Follow-up for this cohort was performed similar to the study group, with routine clinical examinations at 6-month intervals and DUS at 6 and 12 months.

Categorical or discreet count comparison was performed with generation of frequency tables, and Fisher's exact test was reported. Continuous variable comparisons were evaluated with the Wilcoxon rank-sum test. Survival and time-to-event data were analyzed using the Wilcoxon test. Results were considered significant if the p value was less than 0.05.

RESULTS

Preoperative results. There were 15 men (58%) and 11 women (42%) in the study group, with a mean age of 43.2 ± 3.8 years (range, 33 to 49 years) at the time of primary CEA (Table I). Four patients (16%) were less than 40 years old. The mean age of the control group was 69.1 ± 7.4 years (range, 55 to 78 years), with 13 men (43%). Twenty-four of the study patients had neurologic symptoms (92% vs 57%; $p = 0.003$). Demographics, atherosclerotic risk factors and comorbidities, and neurologic presentation for study and control patients are presented in Table I. There was a significantly higher incidence of tobacco abuse (92% vs 70%; $p = 0.036$) among study patients.

Perioperative results. All procedures for study and control patients were performed under general endotracheal anesthesia. Carotid shunting was performed in 31 of 33 CEAs (94% vs 100%), and Dacron patch angioplasty was performed for all primary CEAs (100% vs 94%) in the study group. There were no perioperative (30 days) deaths (0% vs 0%) and no strokes (0% vs 3%; $p = 1.000$) among patients in the study group. There were two perioperative complications (8% vs 3%; $p = 0.592$) of cardiac origin in the study group. The three study patients who underwent secondary procedures for recurrent disease had no postoperative deaths, neurologic events, or other complications.

Late results. Long-term follow-up was available for 23 of 26 study patients (89% vs 100%), with a mean follow-up of 67.4 ± 42.7 months (range, 9 to 143 months), including all patients who underwent secondary procedures (Table II). The mean follow-up for the control group was 57.2 ± 41.5 months (range, 22 to 164 months). Radiographic follow-up with DUS or angiography was available for 26 of 33 (79% vs 90%) of the primary CEAs, with a mean follow-up of 45.2 ± 43.5 months (range, 2 to 118 months), and for all three patients who underwent secondary procedures, with a mean follow-up of 30.3 months (range, 8 to 74 months). For control patients, the mean DUS follow-up was 40.9 ± 38.0

months (range, 5 to 133 months). There was no significant difference in clinical or radiographic follow-up for the study and control groups.

Two late deaths (9% vs 20%; $p = 0.441$) occurred from cerebrovascular accident and myocardial infarction in the study group. Life table analysis (Fig. 1) revealed no significant difference in the 5-year survival rate for young patients compared with control patients (93% vs 81%; $p = 0.373$). However, compared with an age-matched general population, the survival rate for the study group was decreased.

A late ipsilateral stroke occurred in one study patient (4% vs 3%; $p = 1.000$) after carotid occlusion 27 months after staged bilateral carotid repairs. In addition, one patient (4% vs 3%; $p = 1.000$) had a cerebrovascular accident contralateral to the endarterectomized vessel at 34 months. Five study patients (22% vs 17%; $p = 0.730$) experienced recurrent ipsilateral transient ischemic attacks at a mean of 55.5 ± 24.9 months (range, 28 to 100 months) after CEA. DUS revealed moderate restenosis in two of these patients, and their symptoms resolved with medical management. Symptoms developed in the remaining three patients over a mean period of 40.7 ± 21.8 months (range, 16 to 69 months) after the primary CEA. All three patients (11.5% vs 5.7%; $p = 0.665$) had angiographically proved high-grade restenosis and underwent a secondary procedure of carotid resection and polytetrafluoroethylene (PTFE) interposition grafting. After the secondary procedures, there were no recurrent neurologic symptoms at a mean follow-up of 42 ± 22.6 months (range, 27 to 74 months) and no significant restenoses by DUS at a mean follow-up of 30.3 months (range, 8 to 74 months). Life table analysis (Fig. 2) showed no significant difference in ipsilateral symptom-free survival for study patients compared with control patients (5-year, 74% vs. 65%; $p = 0.736$).

Four (17% vs 10%; $p = 0.083$) study patients had contralateral transient ischemic attacks at a mean of 28.2 ± 15.1 months (range, 8 to 48 months) after the primary CEA. Three of these patients had significant stenosis by DUS and angiography. Postoperative surveillance revealed one additional asymptomatic, high-grade contralateral stenosis. CEA was performed for these four patients. Clinical and radiographic follow-up was available for three of the four patients, revealing no recurrent symptoms and one moderate (40% to 70%) restenosis at 18 months for one patient.

Restenosis. DUS for the 26 carotid arteries in the study group showed moderate restenosis (40% to 70%) in two vessels (7.7% vs 2.9%; $p = 0.388$), significant restenosis (>70%) in three vessels (11.5% vs 5.7%; $p = 0.412$), and a total occlusion in two vessels (7.7% vs 5.7%; $p = 0.758$) at a mean follow-up time of 45.2 ± 43.5 months (Table III). The five recurrent stenoses >40% in the study patients were detected at a mean of 33.2 ± 13.4 months (range, 18 to 57 months), and the two occlusions were discovered at 34 and 142 months. Life table analysis (Fig. 3) revealed no significant difference in stenosis-free survival for study patients versus control patients (5-year, 66% vs 60%; $p = 0.378$). Comparison of the six study patients who had seven recurrent stenoses or occlusions with the 17 patients who were without restenoses (Table IV) revealed no significant difference in the incidence of smoking, hypertension, hyperlipidemia, diabetes, coronary artery disease, and female gender.

Table II. Late deaths, strokes, and recurrent disease

	Patients (n = 23)	*Control (n = 30)*	*p*
Mean follow-up (mo)	67.4 ± 42.7	57.2 ± 41.5	0.315
Death	2 (9%)	6 (20%)	0.441
CVA			
Ipsilateral	1 (4%)	1 (3%)	0.848
Contralateral	1 (4%)	1 (3%)	0.848
Recurrent symptoms	5 (22%)	5 (17%)	0.730
Contralateral symptoms	4 (17%)	3 (10%)	0.451
Reoperation	3 (13%)	2 (6%)	0.655
Contralateral CEA	4 (17%)	7 (23%)	1.000

CVA, Cerebrovascular accident.

DISCUSSION

Atherosclerotic carotid occlusive disease in younger patients is uncommon, and few studies have addressed the efficacy of CEA for the prevention of stroke in this population. Some investigators have proposed that the atherosclerotic process seen in these younger patients is more virulent and diffuse than that seen in older populations, with earlier onset, accelerated progression, and higher incidence of recurrent disease after surgical treatment. A higher recurrence rate is well documented for lower extremity occlusive disease in younger patients, with higher failure rates for limb salvage.[5-7] In the few studies that addressed CEA in the young,[3,4,8,9] recurrent disease of the carotid artery also is quite common.

The cause of premature atherosclerosis is not clearly understood but is probably multifactorial in cause. Hypercoagulable states and metabolic abnormalities have been implicated as possible predispos-

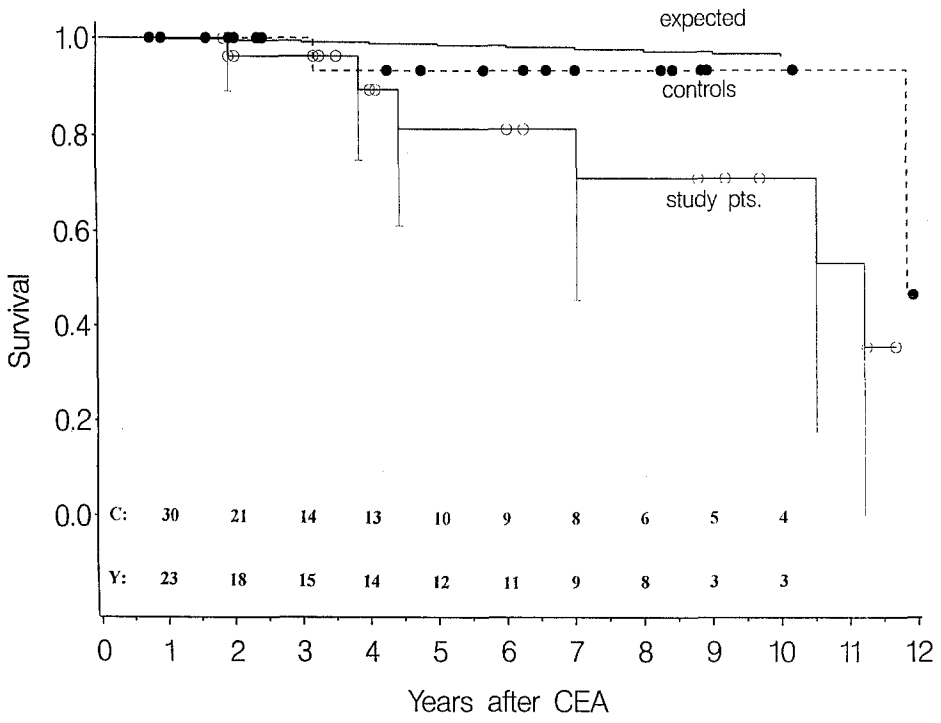

Fig. 1. Kaplan-Meier curves demonstrate survival rates for younger study group patients *(Y)* compared with older control group patients *(C)* and expected survival rates for a 43-year-old general population.[27]

ing factors.[10,11] In addition, clinical risk factors have been identified that are associated with the development of atherosclerosis. Smoking, hypertension, diabetes, and hyperlipidemia are factors most often implicated. In this series a significantly higher incidence of smoking (92% vs 70%; $p = 0.036$) and a trend towards a higher incidence of hyperlipidemia (31% vs 20%; $p = 0.353$) were noted among study patients compared with control patients. Evans et al.[3] noted similar results, finding a significantly higher rate of smoking (91% vs 51%) and hypertension (63% vs 54%) among younger patients.

Young patients with carotid disease were more likely to be symptomatic at presentation in this series (92% vs 55%; $p = 0.003$) with a marked, but not statistically significant, difference in the number (23% vs 7%; $p = 0.080$) who had cerebrovascular accidents before presentation. In a review that compared 42 young patients less than 50 years old with 110 older patients, Valentine et al.[4] also found a significantly higher rate of presenting symptoms in younger patients (96% vs 79%). Higher rates of symptomatic presentation in the young may be related to a lower suspicion of carotid disease and search for asymptomatic bruits or may be a reflection of the more aggressive atherosclerotic process seen in these patients.

Data from this series and others[4] suggest that CEA may be performed safely in this population. In this series, there were no perioperative deaths or strokes in the group of young patients. These results are comparable with or superior to those for older patients, in whom the perioperative mortality rate ranges from 0% to 3%[12-14] and perioperative stroke occurs in 1% to 4%.[12,14] In addition, the rate of perioperative complications was low, with cardiac events accounting for the few complications seen.

The late mortality rate for the study group (9%) was favorable compared with that of the control group (20%) and with that of other series of older patients.[15] The difference in the survival rate between young and old patients in this study at 5 years (93% vs 81%) was not statistically significant. Valentine et al.[4] also found no statistically significant difference in the 5-year survival rate among young (83%) and older (67%) patients after CEA. Although the survival rate for these younger patients is comparable with or better than that for older patients who undergo CEA, it is poor compared with the general age-matched population without atherosclerosis, as

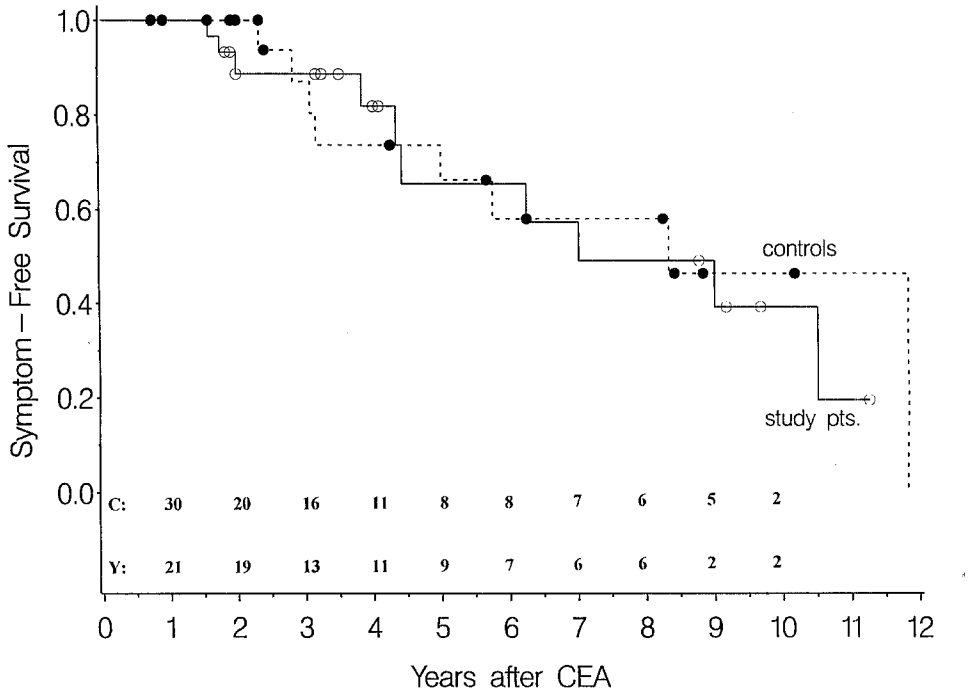

Fig. 2. Kaplan-Meier curves demonstrate symptom-free survival rates for younger study group patients *(Y)* compared with older control group patients *(C)*.

demonstrated in Fig. 1. Evans et al.[3] demonstrated similarly that in patients with premature atherosclerosis who undergo surgery, the survival rate was equivalent to the general population through 5 years, but that by 7 years the survival rate was decreased (79.6% vs 96.4%). The causes of late death in our series, myocardial infarction and stroke, are a reflection of the diffuse vascular disease seen in these patients. Most series of CEA list cardiac events as the leading cause of late death.[12] Cardiac disease is also prevalent in these younger patients with premature atherosclerosis (20% to 43%)[3,4,7,10] and is a major source of late death.

The late stroke rate was comparable for young (4% ipsilateral, 4% contralateral) and older patients (3% ipsilateral, 3% contralateral). The mean follow-up of 67 months for the study group revealed a total late stroke incidence of 8%, with an annual stroke rate of 1.4%. This does not differ significantly from the long-term total stroke incidence of 4.7% to 9% in series of older patients, with annual stroke rates of 1.0% to 1.75%.[12,13,15] The late stroke rate in this series may have been affected by postoperative surveillance and reoperation for those patients in whom significant symptomatic restenosis developed. Three patients with recurrent symptoms and high-grade restenosis underwent secondary procedures and remained without stroke. However, in two patients who had late carotid occlusion and stroke at 34 months and 12 years after CEA, DUS surveillance had been discontinued after normal scans at 12 and 15 months after endarterectomy. Both of these patients had remained neurologically asymptomatic, with no indication of progressive disease.

Table III. Restenosis

	Patients (n = 26)	*Control (n = 35)*	*p*
Total	7 (26.7%)	5 (14.3%)	0.219
40% to 70%	2 (7.7%)	1 (2.9%)	0.570
>70%	3 (11.5%)	2 (5.7%)	0.655
Occluded	2 (7.7%)	2 (5.7%)	1.000
Symptomatic	3 (11.5%)	2 (5.7%)	0.642

The incidence of late recurrent transient ischemic attacks in this series of young patients was also comparable with that of the control cohort (22% vs 17%). In contrast, Valentine et al.[4] found a significantly higher rate of late recurrent neurologic (transient and permanent) symptoms in younger patients compared with older control patients (14% vs 0.9%; $p = 0.002$). It is noted that the incidence of recurrent symptoms in the older control cohort in our series was high compared with that in most series.[12,13] This

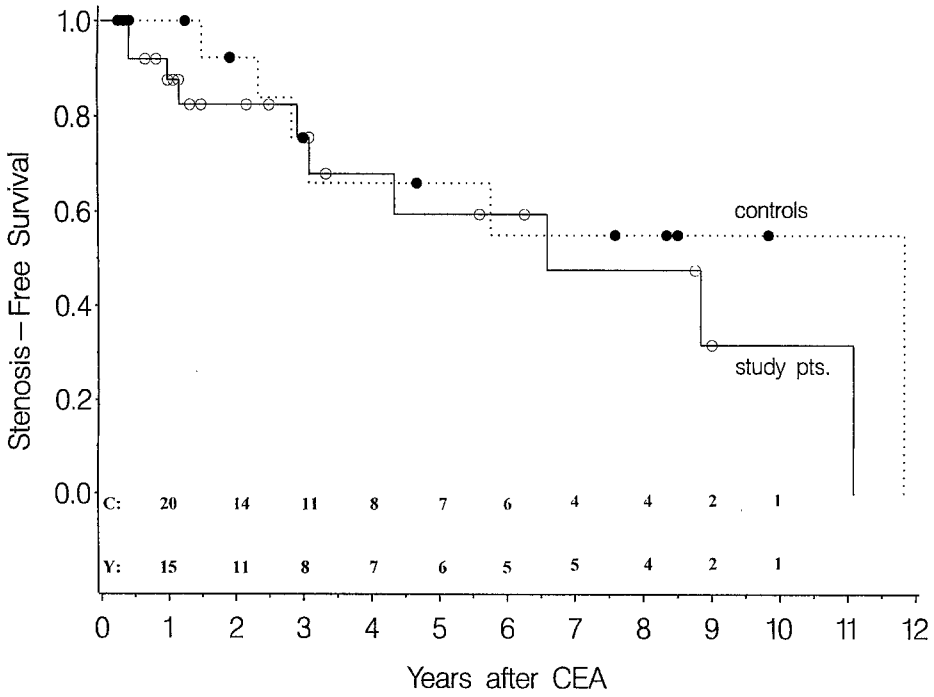

Fig. 3. Kaplan-Meier curves demonstrate stenosis-free survival rates for younger study group patients *(Y)* compared with older control group patients *(C)*.

Table IV. Risk factors for restenosis

	Restenosis (n = 6)	*No restenosis (n = 17)*	*p*
Smoking	100%	86%	1.000
Hypertension	83%	36%	0.141
Hyperlipidemia	33%	21%	0.613
Diabetes	17%	21%	1.000
Coronary disease	50%	29%	0.613
Female	67%	36%	0.336

higher rate of recurrent symptoms seen in the control population may be a reflection of diffuse disease with intracranial atherosclerosis or may be related to the small sample size.

There was a higher rate of restenosis and occlusion among younger patients in our series (26.9% vs 14.3%; $p = 0.219$), but this did not reach statistical significance, possibly a result of the small study size. Others have shown that patients who have premature carotid disease are at higher risk for recurrent stenosis.[4,8,9] The incidence of restenosis in larger series of patients (1.5% to 32%)[16-22] varies depending on the method of detection. However, reoperation rates (1% to 4%),[16-23] generally parallel rates of recurrent neurologic symptoms. The rate of symptomatic restenosis (not including occlusions and strokes) in our series of young patients was three of 23 (11.5% vs 5.7%), whereas the rate of radiographic restenosis was five of 26 vessels (19% vs 9%). The high prevalence of symptomatic restenosis among young patients in this series led to the high rate of reoperation (11.5% vs 5.7%). Other investigators have reported higher rates of reoperation among young patients (6% to 7%) compared with older control groups[3,4] and have noted also a higher rate of contralateral disease development that requires surgery among young patients.

The pathogenesis of carotid restenosis is unknown, and multiple factors may be cited as contributing causes. Biologic factors associated with restenosis, including female gender,[9,22,24,25] continued smoking after primary CEA,[9] and hyperlipidemia[16,17,26] were prevalent in our series and may have contributed to the high rate of restenosis. Although women comprised only 42% of our study population, they accounted for 75% (three of four patients) of the recurrent lesions. In addition, all four patients with restenosis in our series continued to smoke after endarterectomy, and half had hyperlipidemia.

The management of recurrent lesions is controversial because their significance is unclear.[22] The relation between restenosis and recurrent symptoms is not direct. Late (>24 months) recurrent lesions,

particularly when symptomatic, are likely to represent recurrent atherosclerotic disease and should be treated aggressively. Earlier asymptomatic lesions are more likely to represent myointimal hyperplasia, and as many as 20% to 40% of these lesions will regress.[19,22] In our series, the mean time to development of the five restenoses and two occlusions in the study group was 44.6 months, with only one restenosis occurring within 24 months of CEA. Recurrent lesions that were symptomatic and high-grade underwent reoperation, with patients remaining without stroke. Moderate restenoses were early and asymptomatic, consistent with hyperplasia, and were treated medically, also without neurologic sequelae.

The choice of operation for recurrent stenosis generally includes patch angioplasty, with or without endarterectomy, or interposition grafting for cases in which the vessel wall is inadequate for repeat endarterectomy or patch angioplasty. In this series, the secondary procedure in all cases was carotid resection and PTFE interposition grafting. The patency rate of PTFE interposition grafts is good in our experience. There were no symptom recurrences and no significant restenoses for the three patients in this series, who had a mean radiographic follow-up of 31 months.

CONCLUSION

The results of our study suggest that CEA can be performed with a low perioperative morbidity and mortality rate in patients who have premature development of carotid occlusive disease. These patients are frequently smokers and are symptomatic at presentation. However, the rate of recurrent stenosis and symptoms, late stroke, and survival is not significantly different than for older patients who undergo CEA. Aggressive follow-up with serial DUS surveillance for recurrent stenosis and reoperation for significant (>50%) symptomatic restenosis or high-grade asymptomatic restenosis may result in an acceptable late stroke rate in these patients who have aggressive atherosclerotic disease.

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DISCUSSION

Dr. David Rosenthal (Atlanta, Ga.). I would like to congratulate Dr. Martin on his presentation, which I understand is his first at the SAVS and we trust not his last. We have just heard an excellent analysis on a difficult subset of patients, those 50 years and younger, who must undergo CEA. When the Baylor group reports on a 15-year experience, we should all pay attention. It is easy for me to comment on this paper, as I basically agree with everything he just said. I therefore only have an anecdotal observation and a few questions. Despite the excellent perioperative morbidity results just presented, I would like to admonish all of us who operate on younger patients to beware of the so-called young carotid vessels. In these patients, in my experience, it is often difficult to obtain a good endarterectomy plane, which can make the distal end point treacherous. I am not sure whether anyone has the answer as to why this is so, but for lack of a better term, I would call it immaturity of the plaque, which is often soft and fibrous without calcification. I do not routinely patch carotid arteries, but I agree with and would advocate patch angioplasty in these younger patients and certainly women with inherently smaller vessels.

My questions have to do with the postoperative management of these patients. Despite the statistical comparison with your control group, if my addition is correct, almost half of these young patients either had an ipsilateral or contralateral transient ischemic attack, had a stroke, died, or had severe carotid disease during follow-up. This may not be statistically significant, which I anticipate is a result of the small study size, but if I were 45 years old I would find this prognosis frightening. Because we know these patients have an aggressive form of atherosclerosis, how should we be monitoring their cerebrovascular and coronary status? Is postoperative aspirin enough or should we consider the use of ticlopidine or warfarin in this patient population?

Your presentation and manuscript were excellent. I would finally comment that, in my opinion, 50 years old is awfully young.

Dr. Gordon H. Martin. Thank you very much, Dr. Rosenthal. I appreciate your review and your comments. First of all, in terms of the question about how to follow-up these patients that have very virulent and accelerated atherosclerosis, in terms of their cerebrovascular status, given the results of our study and studies performed by other investigators, we believe these patients should undergo routine duplex scans probably every 6 months for the first year, and if they prove to have no evidence of restenosis, just once a year after that.

In regards to how to follow them from a coronary standpoint, I think this series as well as others have shown that many of these patients are at high risk for coronary disease. Many have had previous myocardial infarctions, and the remainder are at high risk for future cardiac events. I think in evaluating these patients for surgery, we should be very aggressive about working up their cardiac status, whether it be with a stress thallium or a stress echo study, to get a baseline for their cardiac status.

The last question had to do with how to manage them from a medical standpoint in terms of whether aspirin was enough or should we use other agents. In our experience, ticlopidine has been met with some complications. These patients have a very high tendency to bleed if they are treated with ticlopidine. They are also at risk for the complications of neutropenia, and so our feeling is that aspirin is sufficient treatment for these patients. In terms of warfarin treatment, we believe that the protective benefit from warfarin would probably require a life-long treatment. As such, that would put these patients at the risk of anticoagulation over many, many years. Again, I do not think warfarin would be necessary for these patients either. Currently, we are treating all of these patients with aspirin alone. Hopefully, more effective alternatives will be available in the future.

Dr. Paul R. Liebman (West Palm Beach, Fla.). I am concerned about putting PTFE in the neck in these young people. The concern would be using a prosthetic in a patient who is hopefully going to be around considerably longer than some of our older patients who have carotid disease. What are the thoughts of your group considering the use of vein in this circumstance?

Dr. Martin. We have used PTFE fairly often. In this series we used it three times, and we are currently working on another report of approximately 30 patients who were also treated with PTFE interposition grafts, and we had no complications among those patients with late infectious

problems of patency. Vein interposition grafts have been shown to be very effective, as well. We have used those from time to time, as well. There appears to be no clear superiority with vein versus PTFE. PTFE is an acceptable alternative in our experience.

Dr. James Haynes (Columbia, S.C.). We have had interest in lower extremity atherosclerosis and ischemic disease in young patients. We discovered the elevated number of these patients with hypercoagulation problems. I rise to ask you two questions. First, when you looked at these patients, did you really study the coagulation factors, particularly the thrombolytic activity, as well as the locked protein A, in these people? We have seen very few people with carotid artery disease in the younger patient population, so I congratulate you on this presentation. As for the second question, have you looked at the lower extremity disease in the same group of patients, those who have had the carotid stenosis at less than 50 years of age?

Dr. Martin. We did not specifically look at hypercoagulable states in these patients in terms of measuring protein C, protein S, antithrombin III, and so forth, but I think the series by Dr. Levy and others have shown that there is a very high incidence of hypercoagulable states in these patients; as many as 90% of them may have some type of detectable defect in their coagulation system. In regards to lower extremities, approximately 50% of our younger patients did have a history of significant lower extremity peripheral vascular disease, a very striking number as well in these relatively young patients.

Dr. Clifford Buckley (Temple, Tex.). I enjoyed your paper very much. I may have misunderstood one of the slides, but it looked as though you reported that 20% of the control patients as well as approximately 20% of the study group of patients had symptomatic cerebrovascular complaints during the follow-up period. That seems high for the control group, in my own experience. I do not believe that 20% of our carotid surgery patients become symptomatic in the follow-up period. That is also high as compared with what is reported in the literature.

Dr. Martin. No, you understood correctly, and I would agree with your interpretation. I think most series show that the incidence of late symptoms runs in the range of 1% to 6%. This result is related to the small sample size and more intracranial disease in these older patients.